Editorials

Too Many Lawyers?

THE *AMA News* (September 16, 1983) reports that the American Bar Association (ABA) is concerned about the increase in the number of lawyers, having estimated that by the end of 1983 there would be 650,000 lawyers in the United States and by the mid-1990s they will number 1 million. The ABA is worried that this rapid increase in the number of lawyers "may lead to a decline in the image of lawyers and more client complaints if lawyers resort to shortcuts or delay tactics to keep their incomes up, according to *U.S. News and World Report.*" There may be others who worry, or ought to be worrying, about the economic impact of such a dramatic increase in the number of lawyers. A more or less comparable percentage increase in the numbers of doctors in more or less the same time frame has caused considerable public discussion about the effect this increase might have on the costs.

A precise breakdown of costs for either the health care system or for the legal system is difficult to come by. At the moment more attention is being given to the costs of health care. Its total costs have been linked to the gross national product and are now said to be more than 10% of it. But it is hard to believe that the total costs of our legal system are not of a similar order of magnitude (even if one chooses to overlook what seem to many to be unconscionable awards in some liability suits), and it is also hard to believe that a rapidly increasing number of lawyers will not have an economic impact upon the total cost of the legal system to the public.

More attention is needed to these issues. The costs of the legal system are surely as important to Americans as is the cost of health care. But the present social and political climate being what it is, it seems unlikely that the legal system and the legal profession will be asked the same kind of questions about costs as are being asked of the health care system and the medical profession. In the meantime there would seem to be many other good reasons to be as worried about there being too many lawyers as is the ABA.

MSMW

The Changing Natural and Surgical History of Abdominal Aortic Aneurysms

THE CAUSE OF ARTERIAL ANEURYSMS, especially aneurysm of the abdominal aorta, is indeed poorly understood, as described elsewhere in this issue by Fortner and Johansen in their comprehensive discussion. Only a relatively small percentage of patients have associated occlusive arteriosclerotic disease of the lower extremities, but the coincidence of abdominal aortic aneurysm with coronary artery disease is high. It is a verifiable fact that more aneurysms have been identified in elderly patients in the past two decades, but whether this represents the accidental discovery because of more abdominal and back x-ray films, more ultrasonic studies and more computed tomographic (CT) scans, or the increased number of persons reaching geriatric age is uncertain.

Surgical results have improved progressively over the past 20 years. This has influenced the decisions concerning prophylactic surgical procedures. Sixty-year-old patients with small 4- to 5-cm aneurysms some years ago were followed till the aneurysms reached 6 to 8 cm, then operation often became urgent in a 70-year-old patient with chronic heart or lung disease, who was a much less satisfactory candidate. Now, with the firm knowledge that current synthetic grafts are extremely durable and infrequently complicated, we are secure in operating on younger patients with smaller aneurysms. If operation becomes necessary in octogenarians, careful anesthetic control and precise cardiac monitoring to prevent underhydration or overhydration have reduced the morbidity in these persons. The greatest cause of perioperative and postoperative morbidity and mortality in aneurysm patients is myocardial infarction. This has led to the current controversy over whether all asymptomatic patients with aneurysm should have coronary angiograms and a prophylactic coronary artery surgical procedure before the aneurysm is resected. There is much to be said for this approach; on the other hand, current statistics do not prove that all patients with asymptomatic carotid bruits should have carotid endarterectomy because the incidence of stroke associated with aneurysm repair is low.

A simple protocol for evaluating any patient older than 50 years seen to have calcium in the abdominal aorta is to obtain a lateral x-ray film. If the anterior wall of the aorta is clearly outlined—even when the posterior aortic wall cannot be seen—measuring the maximum distance from anterior aorta to the vertebral body gives an accurate estimation of the diameter.[1] If the aortic diameter cannot be measured with certainty on lateral x-ray film, then abdominal ultrasonogram or CT scan should be done. If the aorta is greater than 4 cm in outside diameter by any of these measurements, and if after careful medical evaluation the patient has a life expectancy of three to five years, then a prophylactic operation should be seriously considered.

To echo again the findings of Szilagyi and associates,[2] prophylactic surgical repair of abdominal aneu-

rysm by an experienced team at least doubles a patient's life expectancy.

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Mesangial IgA Nephropathies—Steady Progress

ELSEWHERE IN THIS ISSUE is described a case of a patient who had clinical manifestations of a systemic, necrotizing or hypersensitivity vasculitis in a distribution most consistent with Schönlein-Henoch purpura. This diagnosis was established by a renal biopsy specimen, which showed glomerulonephritis with granular deposits of IgA and C3 in the mesangium, associated with mesangial hypercellularity and sclerosis. The patient's renal function failed to improve despite his receiving high-dose steroid therapy, and he subsequently died of other causes. Unfortunately, this course is not uncommon in adults with Schönlein-Henoch purpura who may have rather severe renal disease.[1] Hope that more effective therapy can be developed rests heavily on the premise that a better understanding of the disease mechanisms underlying Schönlein-Henoch purpura and related disorders will lead to less empiric approaches to their treatment. In this syndrome, cautious optimism may be warranted because progress is clearly being made.

Schönlein-Henoch purpura is now viewed as one end of a spectrum of diseases associated with microvascular immune deposits in which IgA is the predominant immunoglobulin seen.[1] The most common of these is IgA nephropathy (Berger's disease) in which clinical manifestations are confined to the kidney. IgA nephropathy is a common cause of "benign" recurrent hematuria, usually presenting immediately following viral upper respiratory tract or gastrointestinal tract infection. There are many reasons to believe that Schönlein-Henoch purpura and IgA nephropathy are probably manifestations of the same disease mechanism, though the causes may differ. Both are characterized by deposition of IgA in the glomerular mesangium and in dermal capillaries of involved and uninvolved skin,[2] and by increases in serum polymeric IgA, IgA-containing immune complexes and IgA-bearing peripheral blood leukocytes.[3-5] The clinical manifestations overlap as well. The occurrence of IgA nephropathy and Schönlein-Henoch purpura has been reported in identical twins,[6] and in patients with IgA nephropathy extrarenal manifestations of Schönlein-Henoch purpura have subsequently developed.[1] Almost certainly studies of the pathogenesis of one of these diseases will apply as well to the other.

How do the mesangial IgA deposits that characterize these diseases develop? The mesangium forms a specialized intercapillary tissue that lies between, and provides structural support to, the filtering capillary loops.[7] Mesangial cells and matrix are separated from the glomerular circulation only by a layer of fenestrated endothelial cells. The mesangium is therefore the principal site of deposition of preformed immune complexes as well as other macromolecular debris that accumulates as a consequence of glomerular sieving function.[7] The mesangial deposits of IgA appear to represent immune complexes containing exogenous antigens and IgA antibody to them.[8] This hypothesis is supported by the increased levels of IgA-containing immune complexes usually present in these diseases when appropriate assays that do not depend on complement fixation are used and the correlation of circulating immune complex levels with clinical manifestations in several patients.[4,9] Mesangial immune complex deposition is directly related to renal immune complex delivery, which is substantially increased if the clearance of immune complexes by the extrarenal mononuclear phagocyte system is impaired.[10,11] As in systemic lupus erythematosus, another renal disease characterized by mesangial immune complex deposits, there is now evidence of impaired mononuclear phagocyte system function in these diseases.[12] Moreover, if the mononuclear phagocyte system imposed by the liver between the portal and systemic circulation is partially bypassed by inducing liver disease or ligating bile ducts, raised serum levels of IgA immune complexes and mesangial IgA deposits result.[13,14] Similar deposits have now been well described in cases of human cirrhosis with portal hypertension and may account for "hepatic glomerulosclerosis."[4] Differences in the capacity of the mononuclear phagocyte system to clear an immune complex load, along with possible differences in size, charge and other factors of circulating immune complexes, may determine why some patients have only mesangial deposits of IgA with mild renal disease while others have more extensive deposits in other systemic capillary beds with the clinical features of Schönlein-Henoch purpura.

If IgA is the antibody component of an immune complex deposited in the mesangium, what antigen is it responding to? This question is almost impossible to answer unless one has some clue regarding what antigens to look for, but clues are emerging. IgA is unique among the immunoglobulins in being derived from two relatively separate sources. Serum IgA is made by plasma cells in spleen and bone marrow, is predominantly monomeric and is 80% to 90% IgA_1, while mucosal IgA is made by plasma cells in the lymphoid systems of the upper respiratory and gastrointestinal tracts, is composed of dimers linked by J chain and contains both IgA_1 and IgA_2 in significant amounts.[15] Initial studies relied heavily on the nature of the IgA subgroups in mesangial deposits to distinguish serum from mucosal IgA and came to differing conclusions.[16,17] However, a consensus is now emerging that the deposits, though